INCIDENCE OF HYPOCALCEMIA FOLLOWING NEAR TOTAL THYROIDECTOMY AND TOTAL THYROIDECTOMY

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CERTIFICATE

This is to certify that this dissertation titled **"INCIDENCE OF HYPOCALCEMIA FOLLOWING NEAR TOTAL THYROIDECTOMY AND TOTAL THYROIDECTOMY"** submitted by **DR.LEON.V.ALEXANDER** to the faculty of General Surgery, The TamilNadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from January 2010 to December 2011.

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DECLARATION

I**, DR.LEON.V.ALEXANDER** solemnly declare that the dissertation titled **"INCIDENCE OF HYPOCALCEMIA FOLLOWING NEAR TOTAL THYROIDECTOMY AND TOTAL THYROIDECTOMY"** has been prepared by me. This is submitted to **The TamilNadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MS degree (Branch I) General Surgery.

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CONTENTS

INTRODUCTION

"*Only the man who is familiar with the art and science of the past is competent to aid in its progress in the future*."[17] So wrote Theodor Billroth of Vienna, one of the founders of modern surgery in the second half of the nineteenth century and a major contributor to the surgery of the thyroid.

The thyroid gland lies in front of the neck and gives rise to goiters, which are often large and unsightly, sometimes obstruct the trachea and esophagus, and may threaten life. For these reasons surgeons have long attempted to provide relief, and operations on the thyroid gland have evolved through the centuries as part and parcel of surgery as a whole. Until about 100 years ago operations were undertaken with trepidation under primitive conditions and often themselves proved fatal. Today they are routine, safe procedures with little morbidity.

Goiters (deriverd from latin word ‘guttur’ = throat) are very common in many parts of the world and were recognized long before the thyroid itself. Thomas Wharton of London described the gland in his Adenographia in 1656 (p. E.2), named it "glandul thyroidoeis," and found that one

specimen weighed six drachms (26 grams). The first credible accounts of thyroid surgery emanated from the writings of Roger Frugardi published at about 1170.[8] The first well-documented partial thyroidectomy was undertaken in 1791by Pierre- Joseph Desault[9]. Guillaume Dupuytren, also of Paris, undertook *total* thyroidectomy in 1808, there was little blood loss, but the patient died from "shock".Dr. Cabaret of St. Malo, who probably performed the first successful total thyroidectomy for a midline goiter, the size of an ostrich egg, with little bleeding.[10] The most notable thyroid surgeons were Emil Theodor Kocher (1841–1917) and C.A. Theodor Billroth (1829–1894), who performed thousands of operations with increasingly successful results. In 1909, Kocher was awarded the Nobel Prize for medicine in recognition "for his works on the physiology, pathology, and surgery of the thyroid gland."[4]Billroth reported 48 thyroidectomies performed since 1877, in which only four patients died,was the first to use artery forceps to prevent and stop hemorrhage. Postoperative tetany was probably first described by Wolfler in 1879 in the first of Billroth's patients to survive total extirpation .[9] One of Billroth's forty-eight patients reported in 1881 died from Hypocalcemic tetany.

The surgical anatomy and blood supply of the parathyroids were studied and described by Halsted in 1907[11]. Hypocalcemia was presumed to be due to inadvertent removal of parathyroid tissue at operation.[12] Hypoparathyroidism could not be accounted for entirely by inadvertent removal of parathyroid tissue, and it was proposed that damage to the vascular pedicle of the gland was a common cause.[13] Another suggestion was that in thyrotoxic patients postoperative hypocalcemia was due to the hunger of osteoporotic bones for calcium,so called 'Hungry bone syndrome'.[14]

Despite the advances that have been made in thyroid surgery with the use of latest equipment and techniques the danger of hypocalcemic tetany is still real and every surgeon should be thorough about the anatomy of thyroid and parathyroid and be ready to deal with its complications . This dire complication represents a major concern for thyroid surgeons as the consequences of chronic hypocalcaemia are often insidious and potentially severe. Permanent hypocalcaemia is a common cause of malpractice litigation after endocrine surgery.

AIMS & OBJECTIVES OF THE STUDY

This study aims to evaluate the incidence of hypocalcemia(both transient and permanent) following near-total thyroidectomy and total thyroidectomy.

This study will include the following;

1. Incidence of hypocalcemia following Total and Near-Total thyroidectomy
2. Follow up of patients upto a period of 6 months.
3. Broad overview of literature about the merits and demerits of surgical procedures used ,early detection and prediction of post-operative hypocalcemia ,treatment of hypocalcemia and measures to prevent its occurrence.

ANATOMY OF THYROID & PARATHYROID GLANDS

THYROID

The great Italian Renaissance anatomist, Andreas Vesalius of Padua, gave the first description of thyroid in De Humani Corporis Fabrica in 1543.[1]The thyroid gland is the biggest gland in the neck[(1)]. The term *thyroid gland* (Greek *thyreoeides*, shield-shaped) is, however, attributed to Thomas Wharton in his *Adenographia* (1656), He believed that it served to lubricate, drain, and warm the larynx. In 1776, the thyroid was classified as a ductless gland by Albrecht von Haller and was thought to have numerous functions ranging from lubrication of the larynx to acting as a reservoir for blood to provide continuous flow to the brain, to beautifying women's necks. Burnt seaweed was considered to be the most effective treatment for goiters by the Chinese from 1600 B.C.[(4)] In 1776, the thyroid was classified as a ductless gland by Albrecht von Haller and was thought to have numerous functions ranging from lubrication of the larynx to acting as a reservoir for blood to provide continuous flow to the brain, to beautifying women's necks.

EMBRYOLOGY

The thyroid gland arises as an outpouching of the primitive foregut around the third week of gestation. It originates at the base of the tongue at the foramen cecum. Endoderm cells in the floor of the pharyngeal anlage thicken to form the medial thyroid anlage that descends in the neck anterior to structures that form the hyoid bone and larynx. During its descent, the anlage remains connected to the foramen cecum via an epithelial-lined tube known as the *thyroglossal duct*. The epithelial cells making up the anlage give rise to the thyroid follicular cells. The paired lateral anlages originate from the fourth branchial pouch and fuse with the median anlage at approximately the fifth week of gestation. The lateral anlages are neuroectodermal in origin (ultimobranchial bodies) and provide the calcitonin producing parafollicular or C cells, which thus come to lie in the superoposterior region of the gland. Thyroid follicles are initially apparent by 8 weeks, and colloid formation begins by the eleventh week of gestation.

General Topography

The thyroid gland consists typically of two lobes, a connecting isthmus, and an ascending pyramidal lobe. One lobe, usually the right, may be smaller than the other (7 percent) or may even be completely absent (1.7 percent). The isthmus is absent in about 10 percent of thyroid glands, and the pyramidal lobe is absent in about 50 percent.

The thyroid gland rests on the anterolateral aspect of the cricothyroid and trachea. The gland itself has a bilobed shape and typically weighs 15–25 g, depending on sex and age. The thyroid lobes extend to the midthyroid cartilage superiorly and lie adjacent to the carotid sheaths and sternocleidomastoid muscles laterally. The strap muscles (sternohyoid, sternothyroid, and superior belly of the omohyoid) are located anteriorly and are innervated by the ansa cervicalis (ansa hypoglossi). The thyroid gland is enveloped by a loosely connecting fascia that is formed from the partition of the deep cervical fascia into anterior and posterior divisions. The true capsule of the thyroid is a thin, densely adherent fibrous layer that sends out septa that invaginate into the gland, forming pseudolobules. The thyroid capsule is

condensed into the posterior suspensory or Berry's ligament near the cricoid cartilage and upper tracheal rings.

Vascular Supply

The thyroid gland competes with the adrenal glands for having the greatest blood supply per gram of tissue.[15] The thyroid is a highly vascular gland that has a redundant arterial supply from the superior thyroid artery (a branch of the external carotid artery) and the inferior thyroid artery (ITA) (a branch of the thyrocervial trunk) with abundant collaterals.[2]

Arteries

Two paired arteries, the superior and inferior thyroid arteries, and an inconstant midline vessel, the thyroid ima artery, supply the thyroid . The superior thyroid artery arises from the external carotid artery just above, at, or just below the bifurcation of the common carotid artery. It passes downward and anteriorly to reach the superior pole of the thyroid gland. In part of its course, the artery parallels the external branch of the superior laryngeal nerve which supplies the cricothyroid muscle and the cricopharyngeus muscle, the lowest voluntary part of the pharyngeal musculature. There are six branches of the superior thyroid artery : the

infrahyoid, sternocleidomastoid, superior laryngeal, cricothyroid, inferior pharyngeal constrictor, and terminal branches of the artery for the blood supply of the thyroid and parathyroid glands. Usually there are two branches to the thyroid —the anterior and posterior— but occasionally there may be a third, the so-called lateral branch. At the superior pole, the superior thyroid artery divides into anterior and posterior branches. The anterior branch anastomoses with the contralateral artery;[108] the posterior branch anastomoses with branches of the inferior thyroid artery. From the posterior branch, a small parathyroid artery passes to the superior parathyroid gland.

The inferior thyroid artery usually arises from the thyrocervical trunk, but in about 15 percent of individuals it arises directly from the subclavian artery. The recurrent laryngeal nerve may pass anterior or posterior to the artery, or between its branches . The lowest branch sends a twig to the inferior parathyroid gland and supplies the lower pole of the thyroid gland. The upper branch supplies the posterior surface of the gland, usually anastomosing with a descending branch of the superior thyroid artery.

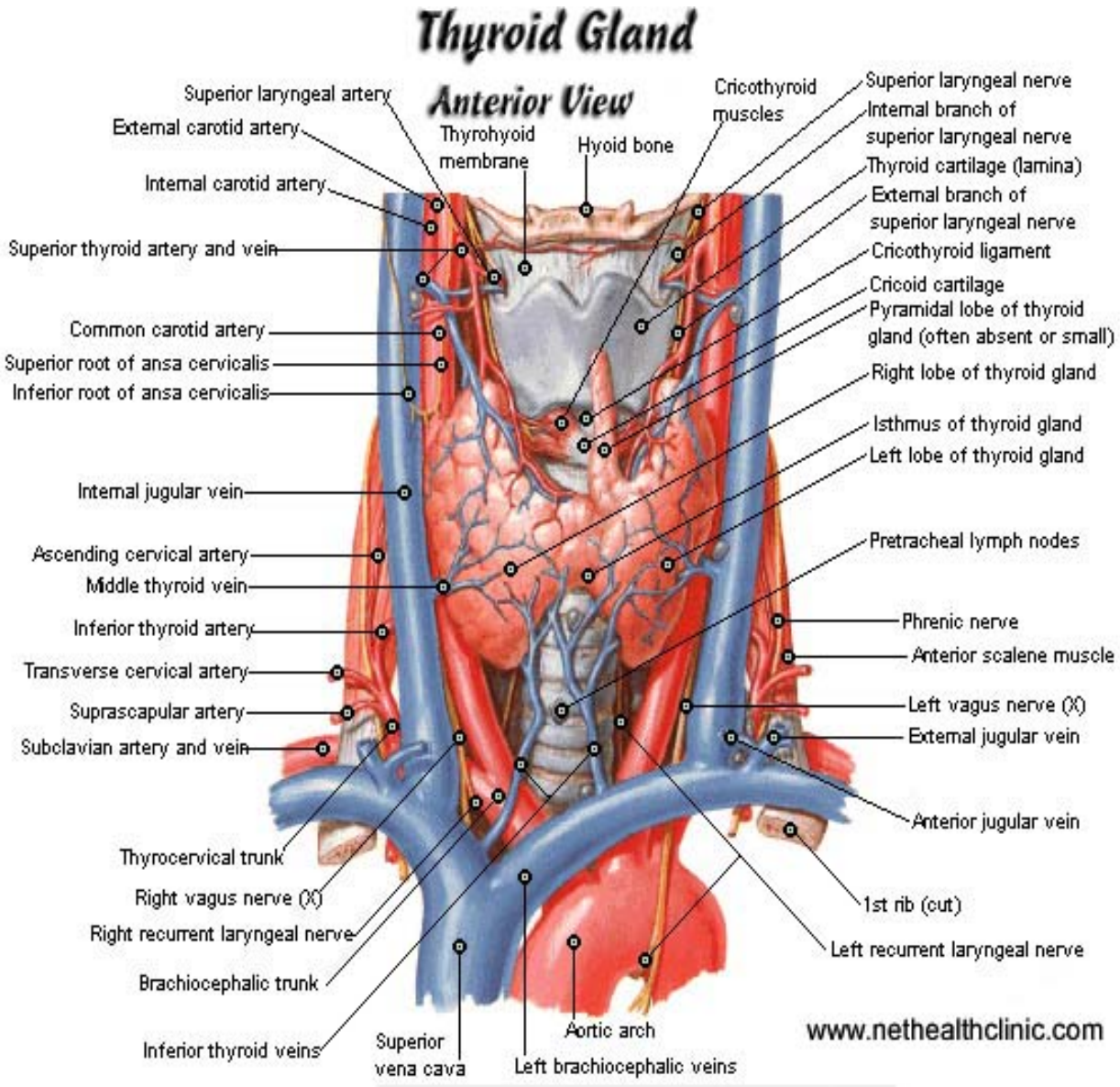
The thyroid ima artery is unpaired and inconstant. It arises from the brachiocephalic artery, the right common carotid artery, or the aortic arch. It

occurs in about 10 percent of individuals, according to Montgomery[16]. Its position anterior to the trachea makes it important in tracheostomy.

Veins

Veins of the thyroid gland form a plexus of vessels lying in the substance and on the surface of the gland. The plexus is drained by three pairs of veins, the superior, middle, and inferior thyroid veins.

The superior thyroid vein accompanies the superior thyroid artery. Emerging from the superior pole of the thyroid, the vein passes superiorly and laterally across the omohyoid muscle and the common carotid artery to enter the internal jugular vein alone or with the common facial vein. The middle thyroid vein arises on the lateral surface of the gland at about two-thirds of its anteroposterior extent. It crosses the common carotid artery to open into the internal jugular vein. The inferior thyroid vein is the largest and most variable of the thyroid veins. The right vein leaves the lower border of the thyroid gland, passes anterior to the brachiocephalic artery, and enters the right brachiocephalic vein. The left vein crosses the trachea to enter the left brachiocephalic vein.


Thyroid Gland
Anterior View
Superior laryngeal artery
External carotid artery
Internal carotid artery
Superior thyroid artery and vein
Common carotid artery
Superior root of ansa cervicalis
Inferior root of ansa cervicalis
Internal jugular vein
Ascending cervical artery
Middle thyroid vein
Inferior thyroid artery
Transverse cervical artery
Suprascapular artery
Subclavian artery and vein
Thyrocervical trunk
Right vagus nerve (X)
Right recurrent laryngeal nerve
Brachiocephalic trunk
Inferior thyroid veins
Superior vena cava
Aortic arch
Left brachiocephalic veins
Thyrohyoid membrane
Hyoid bone
Cricothyroid muscles
Superior laryngeal nerve
Internal branch of superior laryngeal nerve
Thyroid cartilage (lamina)
External branch of superior laryngeal nerve
Cricothyroid ligament
Cricoid cartilage
Pyramidal lobe of thyroid gland (often absent or small)
Right lobe of thyroid gland
Isthmus of thyroid gland
Left lobe of thyroid gland
Pretracheal lymph nodes
Phrenic nerve
Anterior scalene muscle
Left vagus nerve (X)
External jugular vein
Anterior jugular vein
1st rib (cut)
Left recurrent laryngeal nerve
www.nethealthclinic.com

Nerve supply

The left RLN arises from the vagus nerve where it crosses the aortic arch, loops around the ligamentum arteriosum, and ascends medially in the neck within the tracheoesophageal groove. The right RLN arises from the vagus at its crossing with the right subclavian artery. The nerve usually passes posterior to the artery before ascending in the neck, its course being more oblique than the left RLN.

The RLNs innervate all the intrinsic muscles of the larynx, except the cricothyroid muscles, which are innervated by the external laryngeal nerves. Injury to one RLN leads to paralysis of the ipsilateral vocal cord, which comes to lie in the paramedian or the abducted position. The paramedian position results in a normal, but weak voice, whereas the abducted position leads to a hoarse voice and an ineffective cough. Bilateral RLN injury may lead to airway obstruction, necessitating emergency tracheostomy, or loss of voice.

The superior laryngeal nerves also arise from the vagus nerves. After their origin at the base of the skull, these nerves travel along the internal carotid artery and divide into two branches at the level of the hyoid bone.

The internal branch of the superior laryngeal nerve is sensory to the supraglottic larynx. Injury to this nerve is rare in thyroid surgery, but its occurrence may result in aspiration. The external branch of the superior laryngeal nerve lies on the inferior pharyngeal constrictor muscle and descends alongside the superior thyroid vessels before innervating the cricothyroid muscle.

Sympathetic innervation of the thyroid gland is provided by fibers from the superior and middle cervical sympathetic ganglia. The fibers enter the gland with the blood vessels and are vasomotor in action. Parasympathetic fibers are derived from the vagus nerve and reach the gland via branches of the laryngeal nerves.

The thyroid gland contains two separate physiologic endocrine systems: one responsible for the production of the thyroid hormones thyroxine (T4) and triiodothyronine (T3) , and the other responsible for the production of the hormone calcitonin. The functional unit for thyroid hormone production is the thyroid follicle. This is composed of a single layer of cuboidal follicular cells surrounding a central space filled with colloid. The primary function of the thyroid follicle is to make and store thyroid hormones.Para-follicular C cells

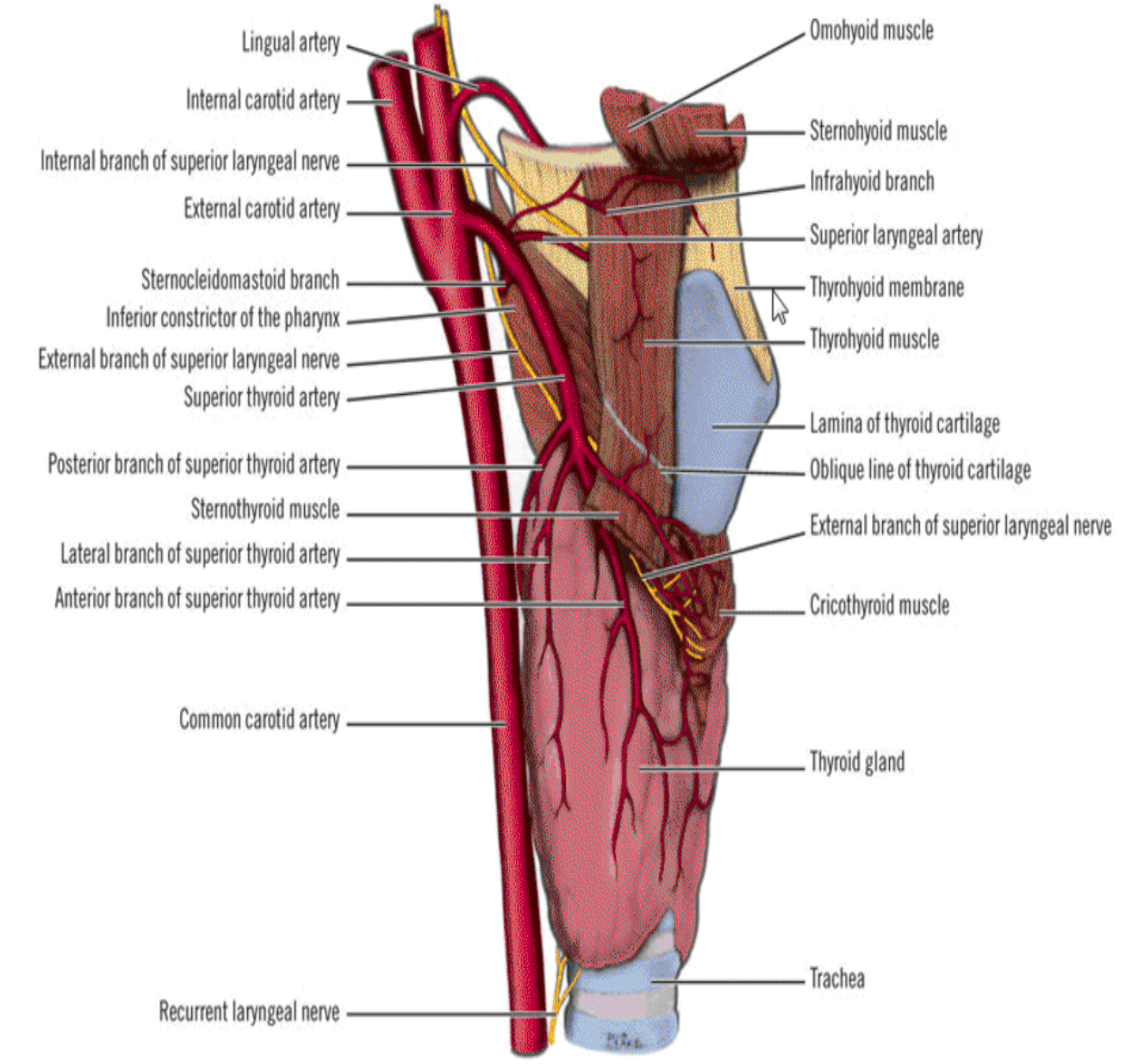


FIG : LATERAL VIEW OF THYROID WITH BLOOD SUPPLY

are responsible for the production of calcitonin which acts in concert with parathyroid hormone (PTH) and vitamin D to regulate serum calcium levels..[3]

PARATHYROIDS

In 1849, the curator of the London Zoological Gardens, Sir Richard Owen, provided the first accurate description of the normal parathyroid gland after autopsy examination of an Indian rhinoceros. However, human parathyroids were not grossly and microscopically described until 1879 by Ivar Sandström, a medical student in Uppsala, Sweden. He suggested that these glands be named the *glandulae parathyroideae*, although their function was not known.

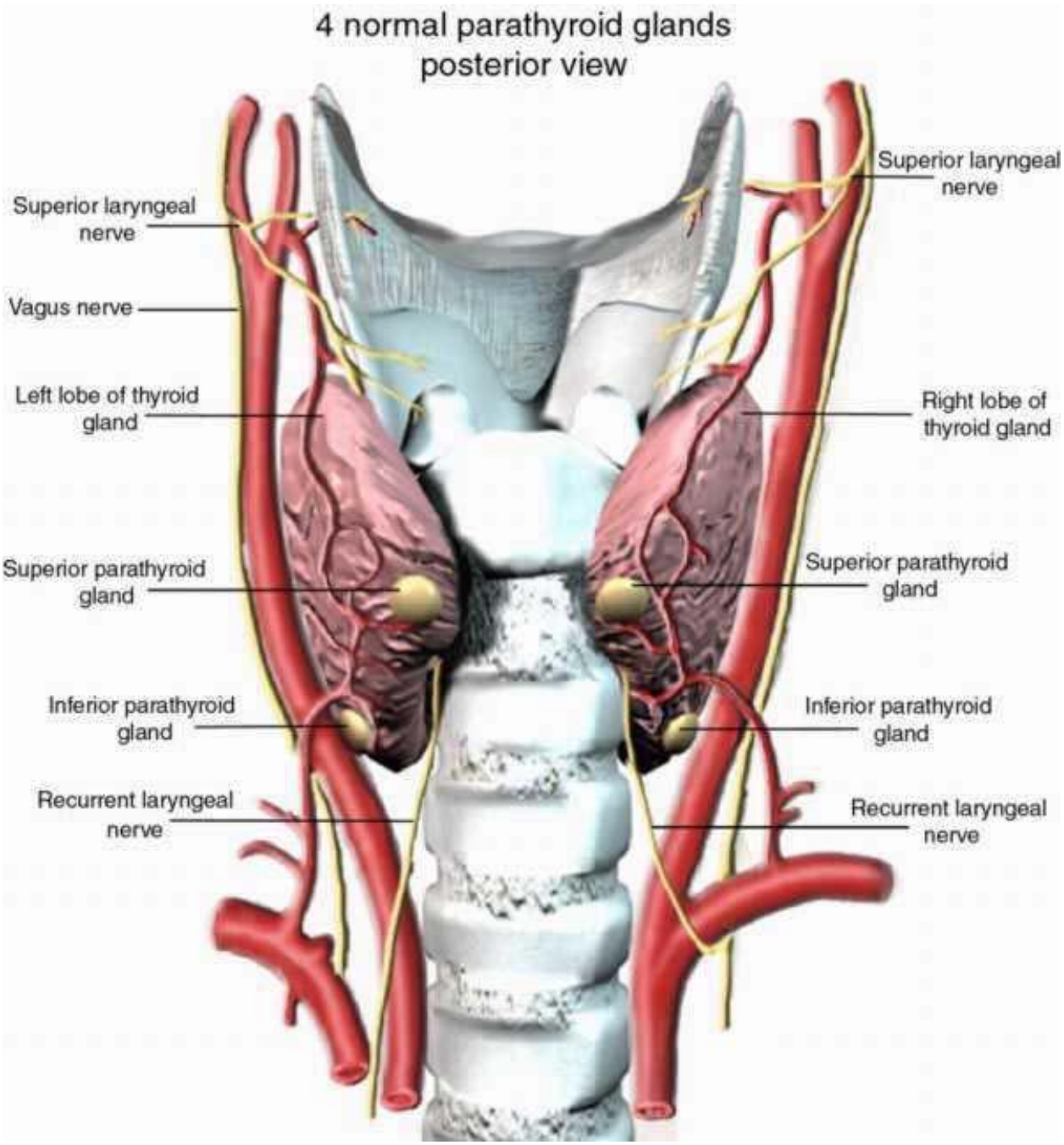
ANATOMY & EMBRYOLOGY

In humans, the superior parathyroid glands are derived from the fourth branchial pouch, which also gives rise to the thyroid gland. The third branchial pouches give rise to the inferior parathyroid glands and the thymus.

The parathyroids remain closely associated with their respective branchial pouch derivatives. The position of normal superior parathyroid glands is more consistent, with 80% of these glands being found near the posterior aspect of the upper and middle thyroid lobes, at the level of the cricoid cartilage. Approximately 1% of normal upper glands may be found in the paraesophageal or retroesophageal space. Enlarged superior glands may descend in the tracheoesophageal groove and come to lie caudal to the inferior glands.[18]

The position of normal superior parathyroid glands is more consistent, with 80% of these glands being found near the posterior aspect of the upper and middle thyroid lobes, at the level of the cricoid cartilage. The position of the inferior glands, however, tends to be more variable due to their longer migratory path. Undescended inferior glands may be found near the skull base, angle of the mandible, or superior to the upper parathyroid glands along with an undescended thymus. The frequency of intrathyroidal glands is about 2%. Supernumerary glands were present in 13% of patients, most commonly in the thymus. Only 3% of patients had less than four glands.[4]

Both the superior and inferior parathyroids are usually supplied by the inferior thyroid artery: 86.1% on the right side, 76.8% on the left. In the


4 normal parathyroid glands
posterior view
Superior laryngeal nerve
Vagus nerve
Left lobe of thyroid gland
Superior parathyroid gland
Inferior parathyroid gland
Recurrent laryngeal nerve
Superior laryngeal nerve
Right lobe of thyroid gland
Superior parathyroid gland
Inferior parathyroid gland
Recurrent laryngeal nerve

absence of an inferior thyroid artery, both the superior and inferior parathyroid glands were supplied by the superior thyroid artery in the majority of cases.[5]

HISTOLOGY

Histologically, parathyroid glands are composed of chief cells and oxyphil cells arranged in trabeculae, within a stroma composed primarily of adipose cells . The parathyroid glands of infants and children are composed mainly of chief cells, which produce parathyroid hormone (PTH). Acidophilic, mitochondria-rich oxyphil cells are derived from chief cells, can be seen around puberty, and increase in numbers in adulthood. A third group of cells, known as *water-clear cells*, also are derived from chief cells, are present in small numbers, and are rich in glycogen. Although most oxyphil and water-clear cells retain the ability to secrete PTH, their functional significance is not known.[4]

Perhaps all the parathyroid cells participate in the secretion of the parathyroid hormone, parathormone (PTH), and in the regulation of calcium and phosphate metabolism. To maintain normal calcium in the blood, a feedback system is formed between the circulating calcium and the secretion of PTH. Too much serum calcium inhibits production of PTH; too little stimulates secretion.[5]

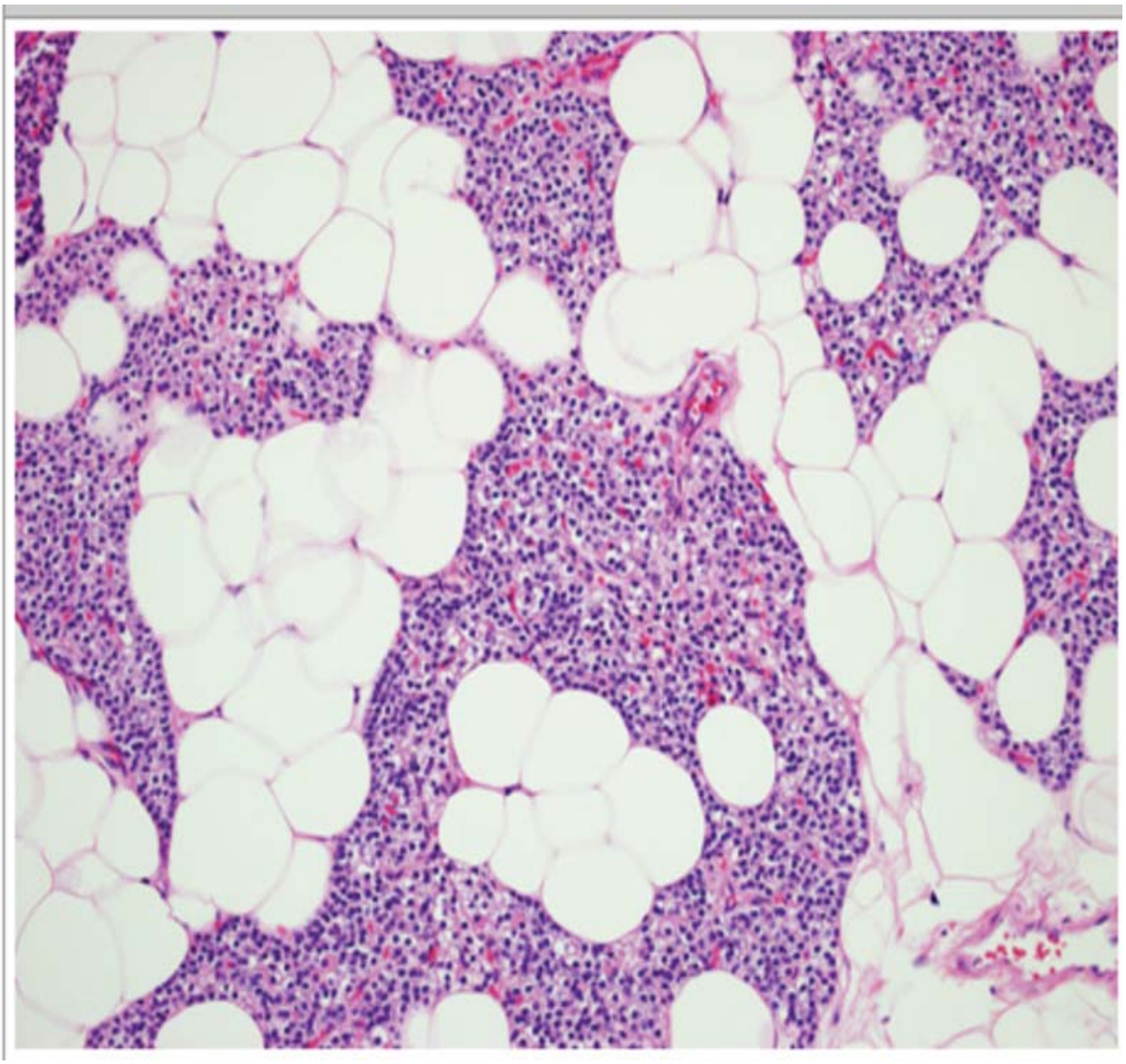


FIG: NORMAL PARATHYROID HISTOLOGY SHOWING CHIEF CELLS INTERSPERSED WITH ADIPOSE CELLS.

PARATHYROID PHSIOLOGY & CALCIUM HOMEOSTASIS

Calcium exists in extracellular plasma in a free ionized state, as well as bound to other molecules. So-called normal plasma levels of total calcium vary between laboratories, but the range of (bound and unbound) calcium is usually between 8.5 and 10.2 mg/dL (2.2 and 2.5 mmol/L). The biologically inert bound fraction (55% of the total) binds to proteins. Changes in albumin alter total calcium levels significantly because the majority of protein-bound calcium associates with albumin (80%). A small percentage of calcium is associated with other proteins, such as β-globulins, or with nonprotein molecules, such as phosphate and citrate. Consequently, ionized calcium levels are measured when required. Forty-five percent of the total calcium is biologically active and exists in the ionized form, with a normal level of 4.5 to 5.0 mg/dL. Ionized calcium levels are inversely affected by the pH of blood; a 1-unit rise in pH will decrease the ionized calcium level by 0.36 mmol/L.[14] Accordingly, patients who are hypocalcemic and hyperventilate can enhance their hypocalcemic symptoms, including perioral paresthesia, tingling in the fingers and toes, muscle cramping, and seizures.

Levels of calcium are highly modulated through a delicate interplay between PTH, calcitonin, and vitamin D acting on target organs such as bone, kidney, and the gastrointestinal tract[6].

Chief cells in the parathyroid glands secrete PTH, an 84–amino acid protein, whenever serum calcium levels fall. PTH binds to its peripheral receptors and stimulates osteoclasts to increase bone resorption, the kidney to increase calcium resorption and renal production of 1,25-dihydroxyvitamin D_3 ($1,25[OH]_2D_3$), and the intestine to increase absorption of calcium and phosphate. Together, these processes raise the serum calcium level. The recently cloned calcium-sensing receptors (CaSRs) in the parathyroid glands detect changes in calcium levels, which results in a negative feedback loop that decreases PTH production.[7]

Parathyroid Hormome(PTH)

The parathyroid cells rely on a G-protein coupled membrane receptor, designated the calcium-sensing receptor (CASR), to regulate PTH secretion by sensing extracellular calcium levels[19]. PTH secretion also is stimulated by low levels of 1,25-dihydroxy vitamin D, catecholamines, and hypomagnesemia. The PTH gene is located on chromosome 11. PTH is

synthesized in the parathyroid gland as a precursor hormone preproPTH, which is cleaved first to pro-PTH and then to the final 84-amino-acid PTH. Secreted PTH has a half-life of 2 to 4 minutes. In the liver, PTH is metabolized into the active N-terminal component and the relatively inactive C-terminal fraction. The C-terminal component is excreted by the kidneys.

PTH functions to regulate calcium levels via its actions on three target organs, the bone, kidney, and gut. PTH increases the resorption of bone by stimulating osteoclasts and promotes the release of calcium and phosphate into the circulation. At the kidney, calcium is primarily absorbed in concert with sodium in the proximal convoluted tubule. PTH acts to limit calcium excretion at the distal convoluted tubule via an active transport mechanism. PTH also inhibits phosphate reabsorption (at the proximal convoluted tubule) and bicarbonate reabsorption. It also inhibits the Na^+/H^+ antiporter, which results in a mild metabolic acidosis in hyperparathyroid states. PTH and hypophosphatemia also enhance 1-hydroxylation of 25-hydroxyvitamin D, which is responsible for its indirect effect of increasing intestinal calcium absorption.[4]

Calcitonin

The Ca^{2+}-lowering hormone has been named **calcitonin.** In nonmammalian vertebrates, the source of calcitonin is the **ultimobranchial bodies,** a pair of glands derived embryologically from the fifth branchial arches. In mammals, these bodies have for the most part become incorporated into the thyroid gland, where the ultimobranchial tissue is distributed around the follicles as the **parafollicular cells,** which are also known as the clear or C cells

Calcitonin is a 32–amino acid protein secreted by the parafollicular cells of the thyroid gland in response to high calcium levels. Measurement of circulating calcitonin by immunoassay indicates that it is not secreted until the plasma calcium level reaches approximately 9.5 mg/dL and that above this calcium level, plasma calcitonin is directly proportionate to plasma calcium. β-Adrenergic agonists, dopamine, and estrogens also stimulate calcitonin secretion. Gastrin, CCK, glucagon, and secretin have all been reported to stimulate calcitonin secretion, with gastrin being the most potent stimulus. Human calcitonin has a half-life of less than 10 minutes.

Its actions oppose those of PTH. Calcitonin rapidly inhibits bone resorption, thereby leading to a transient decrease in serum calcium levels. Calcitonin plays a minimal, if any, role in the regulation of calcium levels in humans. However, it is very useful as a marker of MTC and in treating acute hypercalcemic crisis.[7]

Vitamin D

The active transport of Ca^{2+} and PO_4^{3-} from the intestine is increased by a metabolite of **vitamin D.** The term "vitamin D" is used to refer to a group of closely related sterols produced by the action of ultraviolet light on certain provitamins.

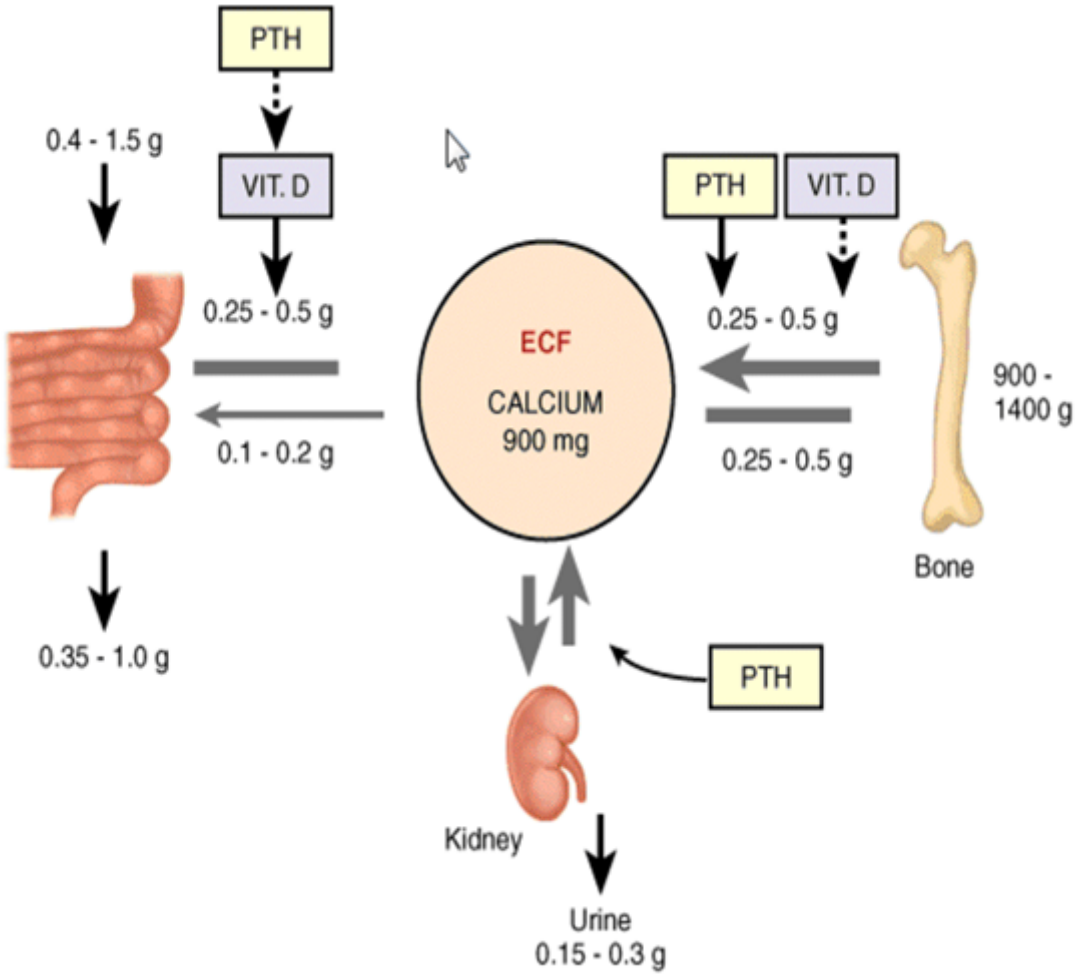
Vitamin D_3, which is also called cholecalciferol, is produced in the skin of mammals from 7-dehydrocholesterol by the action of sunlight. The reaction involves the rapid formation of previtamin D_3, which is then converted more slowly to vitamin D_3. Vitamin D_3 and its hydroxylated derivatives are transported in the plasma bound to a globulin vitamin D-binding protein (DBP). Vitamin D_3 is also ingested in the diet.

Vitamin D refers to vitamin D2 and vitamin D3 , both of which are produced by photolysis of naturally occurring sterol precursors. Vitamin D_2

is available commercially in pharmaceutical preparations, whereas vitamin D_3 is the most important physiologic compound and is produced from 7-dehydrocholesterol, which is found in the skin. Vitamin D is metabolized in the liver to its primary circulating form, 25-hydroxyvitamin D. Further hydroxylation in the kidney results in 1,25-dihydroxy vitamin D, which is the most metabolically active form of vitamin D. Vitamin D stimulates the absorption of calcium and phosphate from the gut and the resorption of calcium from the bone.

Summary

The actions of the three principal hormones that regulate the plasma concentration of Ca^{2+} can now be summarized. PTH increases plasma Ca^{2+} by mobilizing this ion from bone. It increases Ca^{2+} reabsorption in the kidney, but this may be offset by the increase in filtered Ca^{2+}. It also increases the formation of 1,25-dihydroxycholecalciferol. 1,25-Dihydroxycholecalciferol increases Ca^{2+} absorption from the intestine and increases Ca^{2+}reabsorption in the kidneys. Calcitonin inhibits bone resorption and increases the amount of Ca^{2+} in the urine.



CALCIUM BALANCE AND FLUXES IN A NORMAL HUMAN.

Solid arrows depict a direct effect, whereas *dashed arrows* depict an indirect effect. The thickness of the arrows is representative of the magnitude of the flux. ECF = extracellular fluid; PTH = parathyroid hormone; VIT. = vitamin

SIGNS & SYMPTOMS OF POST THYROIDECTOMY HYPOCALCEMIA

Hypoparathyroidism after thyroidectomy remains the most widely accepted cause of hypocalcaemia[20]. Edis in 1979 reported that injury, devascularization or inadvertent removal of parathyroid tissue must be the first practical consideration in development of hypocalcaemia.[21]

Ligation of the main trunk of inferior thyroid artery lateral in the neck during thyroidectomy gives good haemostasis and avoids injury to recurrent laryngeal nerves, but also it may interfere with the blood supply of parathyroids giving rise to hypoparathyroidism with subsequent hypocalcaemia.[20]

SYMPTOMS

- circumoral and fingertip numbness and tingling
- Mental symptoms - anxiety, confusion, and depression
- Muscle cramps
- Convulsions

SIGNS

- *Chvostek sign*- which is contraction of the facial muscles after tapping on the facial nerve anterior to the ear. Approximately 15% of normal individuals have a positive Chvostek sign, however.[7]
- *Trousseau's sign* -carpopedal spasm which is elicited by occluding blood flow to the forearm after inflation of a blood pressure cuff to 20 mm Hg above the patient's systolic blood pressure for for 2 to 3 minutes.
- Tetany- which is characterized by tonic-clonic seizures, carpopedal spasm, and laryngeal stridor & spasm and bronchospasm.
- Prolongation of QT interval on ECG.
- Contraction at the oral angle is seen in 10% - 25% of the population – good to test pre operatively[76]

CHRONIC COMPLICATIONS OF HYPOCALCEMIA

Chronic complications are intracranial calcifications, particularly of the basal ganglia, various mental disturbances, such as irritability, depression, and even psychosis. Papilledema and other signs of increased intracranial pressure have been reported.

Chronic hypocalcemia may lead to cataract formation, abnormalities of the skin, nails and hair, candida infections and dermatology anomalies. Cardiac effects of hypocalcemia include prolongation of the QT interval and, rarely, congestive heart failure. Dental anomalies depend on the age at onset; in children hypocalcemia can cause enamel hypoplasia and failure of the adult teeth to erupt.

FIG:TROUSSEAU'S SIGN IN A PATIENT WITH HYPOCALCEMIC TETANY *(A.K.A "ACCOUCHER'S HAND")*

REVIEW OF LITERATURE

Definition of Post-thyroidectomy hypocalcemia

In review of literature there are different definitions of post thyroidectomy hypocalcemia mostly based on total serum calcium levels.[23-28]

"Postoperative hypocalcemia(hypoparathyroidism) was defined by postoperative Total serum Ca levels of less than 7.6mg/dl (normal range 8.4-10.4 mg/dl) with or without clinical symptoms of hypocalcemia (neuromuscular irritability including paresthesia, muscle cramps, tetany, or seizures) or subnormal serum Ca levels(7.6-8.4 mg/dl) with neuromuscular symptoms during the first 4 postoperative days."[22]

In our study we have taken a **total serum calcium less than 8.5mg/dl** arbitrarily as a **cut off point for post-operative hypocalcemia.**

Vocal cord paralysis, hemorrhage and hypocalcemia are the most common complications of thyroid and parathyroid surgery Postsurgical

hypocalcemia is usually manifest within the first 24 to 48 hours following surgery. Generally, calcium values and parathyroid responsiveness improve within 1 week after surgery. [3]

If the pre-existing hyperparathyroidism resulted in excessive bone resorption, then a more severe, protracted form of postparathyroidectomy hypocalcemia referred to as "hungry bones" may ensue. "**Hungry bone syndrome"** is typically associated with more severe, symptomatic hypocalcemia and may require treatment for several weeks to months before biochemical parameters normalize. An elevated serum PTH level distinguishes hypocalcemia of hungry bones from other forms of post-parathyroidectomy hypocalcemia.[3]

ETIOLOGY

Etiology of postoperative hypocalcemia has not been definitively elucidated. However, several mechanisms have been proposed[29],

1) Secondary to trauma which usually results from intentional or inadvertent extirpation of the parathyroid glands during thyroidectomy.[3]

2) From interruption of the blood supply to the glands with subsequent infarction
3) Calciuretic effect of intraoperative fluid administration(transient per-operative hemodilution) [29]
4) Decreased parathormone secretion from atrophic glands.[30]
5) "Hungry bone syndrome" related to remineralization of the skeleton.[31]
6) Destruction of the parathyroid glands as a result of lymphadenectomy along the recurrent laryngeal nerve (RLN)[32]
7) Hypoparathyroidism due to hematoma formation.[33]
8) increased release of calcitonin as a result of thyroid manipulation.[77]

PREDICTORS OF POST-OP HYPOCALCEMIA

Clinical and biochemically relevant predictive factors for the development of postoperative transient or permanent hypoparathyroidism, as well as how best to time its prediction, constitute a controversial topic in the literature.[22]

Some studies describe decreasing sCa levels within the first 48 hours after surgery as a safe predictor of postoperative hypoparathyroidism.[34,35,36] According to the study by Reza et al the following are predictors for post-op hypocalcemia[22];

(1.)Total serum Ca Levels

Predicting hypoparathyroidism on the first postoperative day by measuring total Serum calcium levels showed a sensitivity of 18.6% with a specificity of 96.1%. On the second postoperative day, the sensitivity of Serum calcium level measurement rose to 62.8%, with a specificity of 92.9%. The **highest value of total** Serum calcium measurement **to predict hypoparathyroidism** was documented on **the third postoperative day with a sensitivity of 72.1%, a specificity of 92.9%, a PPV of 77.5%, and an NPV of 90.8%.**

(2.) Serum Calcium slope

Serum calcium levels were plotted as a function of time and the slope was defined as an increase or decrease in Serum calcium levels during 2 consecutive measurements within 48 hours after operation (within 24 hours and on the second postoperative day).Serum Ca slope is calculated using the

following equation to evaluate its predictive value for postoperative normocalcemia or hypocalcemia;

Slope=[(Calcium Level 2−Calcium Level 1)/Calcium Level 1]/(Time 2−Time 1)

A positive Serum calcium slope, strongly predictive of a stable postoperative Serum calcium level, showed a sensitivity of 88.4%, a specificity of 35.4% for Postoperative hypocalcemia.

(3.) Intact (i)PTH Level

The sensitivity of iPTH levels as the only predictive value for hypoparathyroidism on the first postoperative day (defining hypoparathyroidism with iPTH levels of 15 pg/mL or less) was 97.7%, with a specificity of 82.6%. The same sensitivity (97.7%) could be documented for the iPTH levels measured on the fourth postoperative day.

(4.) Combination of Serum calcium and Intact iPTH Levels

Using the combined interpretation, we observed the best result with iPTH values (definition of hypoparathyroidism, an iPTH level of <15 pg/mL)

measured on the first postoperative day and Serum calcium values (definition of hypoparathyroidism, Serum calcium level of<7.6mg/dl) measured on the second postoperative day.

The most reliable predictor for determining transient or permanent hypoparathyroidism may be intraoperative iPTH measurements.Finally Reza et al concluded that **measuring iPTH levels 24 hours after total thyroidectomy in combination with Serum calcium levels on the second postoperative day** allows the prediction of hypoparathyroidism with a high sensitivity, specificity, and positive predictive value.[22]

(5.)Total free calcium(ionized Calcium level)[77]

Ionized calcium represents 50% of the total calcium and is the biologically important fraction that is highly regulated. The ionized calcium is calculated by assuming that the albumin concentration is 40 g/L. It is measured by total calcium concentration minus (measured albumin in g/L minus 40), multiplied by 0.02.[77]

(6.) Other factors

According to a study by Yesim et al preoperative serum 25-Hydroxy vitamin-D3 level less than 15 ng/mL, a postoperative serum PTH level less

than 10 pg/mL, and Age older than 50 years were significantly predictive factors for postoperative hypocalcemia.[78]

REVIEW OF OTHER STUDIES

Although Lindblom et al found no overall significant difference between measurements of intact iPTH levels and measurements of Serum calcium concentrations on the first postoperative day for predicting long-term hypoparathyroidism, monitoring of intact iPTH levels could predict which patients may need intravenous calcium supplementation during the first 24 hours postoperatively.[23]

There is ***considerable controversy in the literature regarding the utility of intact iPTHmeasurement*** for prediction of postoperative hypocalcemia in patients undergoing surgery for primary hyperparathyroidism. Warren et al. concluded that intact iPTH levels > 15 pg/ml after total thyroidectomy indicate a low risk of postoperative hypocalcemia.[37]

However most studies recommending intraoperative monitoring of iPTH levels, levels that were less below the normal range at the end of or

immediately after the operation were highly correlative with postoperative hypoparathyroidism and seemed to allow for early prediction, with a sensitivity and specificity ranging from 71% to 100%.[23,41-43]

In addition, Higgins et al. concluded from their findings that intraoperative PTH monitoring is useful in predicting postoperative hypocalcemia following total thyroidectomy.[42]

In the study of Elaraj and co-researchers iPTH monitoring was able to predict postoperative hypocalcemia, but this correlation was significant only in multiglandular disease and **not in patients with a single adenoma**[38]

Lo et al. conducted a study on the applicability of intraoperative hormone assay during thyroidectomy and concluded that iPTH monitoring during thyroidectomy enables the identification of patients at risk to develop postoperative hypocalcemia.[39] In the study by Quiros and colleagues ,72 patients undergoing thyroid surgery underwent an Intra-operative Quick-PTH monitoring, and the authors concluded that an Intra operative-Quick

PTH level < 10 pg/ml is a strong predictor of hypoparathyroidism after thyroid surgery[40].

Some authors ascribe a high predictive value to **intraoperative or early perioperative measurement of intact parathyroid hormone levels (iPTH)** challenging the common practice of assessing serum calcium (sCa) levels daily until an increase is observed.[23,41-44] Intra and postoperative intact parathyroid hormones has been embraced with enthusiasm by many surgeons as a means to detect patients with the highest risk of severe hypocalcemia, but its **major limitation for wider clinical use is the cost factor.**

However monitoring of iPTH levels during thyroidectomy may be easily organized in centers of endocrine (thyroid) surgery. However, most patients with thyroid problems are treated in smaller surgical units. Therefore, **monitoring of intraoperative iPTH** levels may be a **logistical problem and increase the costs.[39,41,44]**

Others have suggested that Serum calcium concentrations should be measured only in selected patients[45] or during the initial 24-hour postoperative period.[34,35,36]

However decreased total Serum calcium levels measured on postoperative day 1 were observed in 97.6%. This phenomenon has frequently been debated and often partially explained by hemodilution after surgery .[46.47]

Certain authors recommend measurement of free/ionized calcium levels in the assesment of post thyroidectomy patients(*Faiza A Qari et al*[77]*)* because the fall in serum total protein level due to hemodilution associated with the stress of surgery causes a **decrease in serum total calcium level unrelated to parathyroid** function.[90.91]

Therefore, on the first 2 postoperative days, **interpretation of intact iPTH levels in combination with serum Calcium levels** seems an **optimal strategy** for predicting patients at risk of hypoparathyroidism and hypocalcemia.[27,41,44]

MANAGEMENT OF POST-OP HYPOCALCEMIA

If the patient is asymptomatic with **mild hypocalcemia** (7.5 to 8.5 mg/dL), oral therapy consisting of up to 1000 mg of elemental calcium every 6 hours and 0.25 to 2.0 ug/day of calcitriol [I,25-(OH)z] vitamin D3 should be instituted. As with intravenous therapy, the oral doses can be titrated to keep the calcium in the low-normal range. Magnesium levels should be monitored and repleted because hypomagnesemia results in both impaired PTH secretion and PTH resistance.

In **severe cases** (calcium level < 7.5 mg/dL or severe symptoms), intravenous administration of calcium salts is required. A recommended approach includes using 10 ampules of calcium gluconate (90 mg of elemental calcium per lO-mL ampule) in I L of 5% dextrose with an initial infusion rate of 100 mL/hour. With frequent monitoring of serum calcium levels (usually every I to 2 hours), the infusion rate is titrated to keep the calcium level in the low-normal range. Once stabilized, the patient may be converted to a regimen of oral calcium and calcitriol as described earlier [(4)]

Regarding the duration of post-operative calcium supplementation,a review of literature gives varying time periods with a study by Kanis et al recommending a duration of supplementation of **oral calcium with or without vitamin D_3 for 3 to 6 months**[80].

Another study by Faiza et al supports this duration of post-operative supplementation for 3-6 months.[77]

Unnecessary supplementation may cause hypercalcaemia and hypercalciuria which could results in renal failure.[81]

According to a study by Jong et al routine administration of a supplement containing oral calcium and vitamin D is effective in reducing the incidence and severity of hypocalcemia after total thyroidectomy. In this study, oral calcium (3 g) and vitamin D (1 g) were administered daily to patients for 2 weeks after total thyroidectomy. Given that serum calcium decreased after surgery and then recovered during the postoperative period of more than 1 month in this study, **a treatment duration of 2 weeks** is proposed.

In a study by Moore et al patients were given oral calcium on an empirical basis, an elderly patient supplemented with calcium 5 g/d for 2 weeks became lethargic and hypercalcemic. Moore has recommended that all patients having bilateral thyroid surgery, including total thyroidectomy, should have routine calcium supplementation prior to discharge from the hospital.[84]

We therefore advocate routine oral calcium and vitamin D supplements in the prevention of postoperative hypocalcemia and for increasing the likelihood of a safe and early discharge from the hospital. This will ultimately lead to improved patient satisfaction and significant cost savings.

MEASURES TO PREVENT POST THROIDECTOMY HYPOCALCEMIA

When evaluating the patient prior to surgery, make sure to keep in mind risk factors for postop hypocalcemia,[76]

- Hyperthyroidism
- Large goiters
- Preoperative low serum vitamin D (low sunlight, alcoholism etc)
- Planned level 6 neck dissection, extensive cancer

Permanent hypocalcaemia can be avoided by keeping the plane of dissection near the capsule of thyroid gland to prevent, if possible, removal of parathyroid glands.[79]

Ligation of the inferior thyroid artery branches in the thyroid capsule should be favored over central ligation to preserve vascular supply to the parathyroids.[79]

Surgeons should look in the thyroid specimen for parathyroid glands that may have been removed and re-implant them. To avoid permanent hypoparathyroidism, autotransplantation of parathyroid glands is recommended.[48,49]

Zedenius et al25 found that, after routinely transplanting at least 1 parathyroid gland into the sternocleidomastoid muscle, none of their 100 consecutive patients undergoing total thyroidectomy developed permanent hypoparathyroidism. A similar experience was reported by Lo and Lam.[27,28]

However Parathyroid autotransplantation does not provide absolute protection against permanent hypoparathyroidism. [32,42]

MATERIALS AND METHODS

This study was carried out in 120 patients who underwent Near-Total thyroidectomy and Total thyroidectomy in the department of general surgery,Government Rajaji Hospital,Madurai during the period from 2010 January to December 2011.

This study which was undertaken was a prospective case series study and was started after getting due clearance from the Institute of Ethical committee(IEC),Government Rajaji Hospital.Madurai Medical College.Patients were included in this study after getting their informed written consent.

Inclusion criteria for patients in this study consist of -Patients of both sex and any age presenting with enlargement of both lobes of thyroid (includes both benign and malignant enlargement) proved by ultrasound (US) to be nodular & by laboratory investigations (Thyroid function test) to be euthyroid.

Patients excluded were those with contra-indications for surgery and general anaesthesia and those who were not in a euthyroid state.

Data on patient's demographics, operative aspects, postoperative recovery, complications (post thyroidectomy hypocalemia) and results were collected.

Patients in this study underwent an pre-operative assesment to confirm the diagnosis which included,

(1)Full clinical History

(2)Clinical examination

(3) Fine Needle Aspiration Cytology

(4) Thyroid function test (TFT)

(5) Ultrasound Neck

(6) Indirect laryngoscopy to assess vocal cord status.

All patients included in this study were to be followed up for period of 6 months.

Postoperative assessment will include the following:

- **Immediate post-op period:** in the hospital looking for numbness and other manifestations of hypocalcaemia along with **serum calcium*** on the **second post-op** day.
- **after one month** :serum calcium*
- **after six months** : serum calcium levels*

* In our study a **free/ionised calcium assay** was **not done** due to non availability of this test in our institution and in private labs in madurai .

SURGICAL STRATEGY

In this study two types of surgery were used namely Near total thyroidectomy and Total thyroidectomy,however our **aim is not to compare both techniques** as the former surgery was used in majority of cases in our department.

TOTAL THYROIDECTOMY

Total Thyroidectomy is complete removal of all thyroid tissue without leaving behind any remanant.In our Hospital (Govt.Rajaji Hospital) we currently use a total thyroidectomy only in cases suspicious of malignancy and proven cases of Carcinoma Thyroid.

Review of literature about this procedure is controversial as there are many divergent views about the indications of this procedure.In a recent editorial,Wheeler stated that "total thyroidectomy for benign thyroid disease is likely to become an accepted component of the endocrine surgeon's armamentarium." [50]

Although there has been increasing acceptance for performing total-thyroidectomy for well-differentiated thyroid carcinoma, surgeons still continue to debate whether the potential benefits outweigh the potential complications. There are still some who argue that total thyroidectomy is an operation that is almost never justified.The use of total thyroidectomy for benign thyroid disease is there for even more controversial, although there are increasing numbers of reports recommending its use for bilateral benign multinodular goiter.[52-54]

The only real argument against total thyroidectomy is the potential for an increased risk of complications. However, there is good evidence to show that with increasing experience, the use of appropriate surgical technique, and, most important, the quality training of surgical residents, total thyroidectomy can be performed with minimal complications.[55,56]

In a study by Delbridge et al, the incidence of permanent recurrent laryngeal nerve palsy after total thyroidectomy was only 0.5% , the incidence of permanenthypoparathyroidism and hypocalcemia was only 0.4%. Interestingly, this incidence was little different from the complication rates

recorded after subtotal thyroidectomy (0.2% for permanent recurrent laryngeal nerve palsy and 0.2% for permanent hypoparathyroidism). There is no question that hemostasis is more assured after total thyroidectomy when all vessels have been individually ligated than when dealing with an intact mass of residual vascularized thyroid tissue. parathyroid viability can be just as readily assured by a policy of aggressive parathyroid autotransplantation . However,temporary hypocalcemia remains a treatment problem after total thyroidectomy.[57]

Subtotal thyroidectomy was considered safe compared to total thyroidectomy [59], and it was thought that leaving behind some thyroid tissue would prevent thyroxine supplementaton With long follow-up of these cases, however, it was observed that nodular goiter often recurs after surgery, and the recurrence rate increases with time .[60]

In a study by Reeves et al Subtotal thyroidectomy may also lead to recurrent goiter in up to 23% of patients.[58]Another study by Rojdmark and Jarhult noted a 42% recurrence rate after 30 years of follow-up in a group of

43 patients with multinodular goiter on whom subtotal thyroidectomy had been performed.[61]

Postoperative thyroxine suppression is advocated to prevent goiter recurrence. Failure of thyroxine suppression to prevent recurrence has been observed by many authors.[62,63,64]

There is another issue of underdiagnosed malignancy in casesof multinodular goiter and Graves' disease. Subtotal thyroidectomyin this case represents inadequate surgery.[65,71]

Several studies have shown that total thyroidectomy can be performed with a morbidity rate comparable to that of less radical resections.[66,67,68]Reeve et al. considered it a preferred option, as reoperation for recurrent goiter is associated with increased morbidity.[58,69] Others studies have supported that view.[54,70]

Thus considering all the facts, total thyroidectomy represents the most logical and effective means of treating this disorder, provided the operation is

performed by experienced surgeons who have special interest in endocrine surgery.[69,71]

Currently in our deparment in majority of cases a near-total thyroidectomy is done and total thyroidectomy reserved for cases suspicious and proven for malignancy and is usually combined with a modified radical neck dissection depending on the nodal status.

NEAR TOTAL THYROIDECTOMY

Near-total thyroidectomy is defined as performing a total lobectomy on the dominant side and a subtotal lobectomy on the contralateral side.[57]It consisted of total lobectomy in the lobe having the dominant nodule, with isthmectomy and sub-total lobectomy in the contralateral side, leaving a small quantity of about 2 g of thyroid tissue adjacent to the parathyroid glands and their blood supply.[72]

When the disorder affects both lobes, total or near-total thyroidectomy is mandatory, especially in younger persons, to obviate suppressive therapy and possible relapse.[72]

Subtotal thyroidectomy may be the best elective procedure in older patients to avoid total and permanent dependence on drugs.[73]

Ambrosi et al found that recurrence was inversely related to the extent of resection.[74]

The indications for total thyroidectomy for managing benign thyroid disorders are not well defined, in fact, they are evolving.[54,58,70]

An advantage of near-total thyroidectomy over subtotal thyroidectomy is that the thyroid remnant of about 2 g renders it accessible to 131I ablation if cancer is found in the specimen and obviates reoperation for completion thyroidectomy.[75]

Permanent or temporary hypoparathyroidism is a well-known complication of total thyroidectomy. Excision of parathyroid glands and vascular insufficiency are the main causes of this complication. Identification of the parathyroid glands and meticulous surgical technique to preserve parathyroid circulation are essential during near-total thyroidectomy which were done by consultants or under their guidance in my study.

So currently in our Department we prefer to use near- total thyroidectomy over total thyroidectomy for treatment of benign enlargement of both lobes of thyroid.

FIG: A CASE OF CARCINOMA THYROID WITH BONE SECONDARIES (STERNUM)

FIG: NEAR TOTAL THYROIDECTOMY SPECCIMEN

OBSERVATION & RESULTS

The findings of the study can be tabulated as follows.

TABLE-I

AGE DISTRIBUTION IN THE STUDY

Age in years	**COLLOID GOITRE**	**MNG**	**TOXIC GOITRE**	**CARCINOMA THYROID**
10-20	3	3	-	-
20-30	-	14	1	5
30-40	2	20	6	7
40-50	1	22	4	6
>50	1	13	4	8

TABLE-II

SEX DISTRIBUTION IN THE STUDY

SEX	**MNG**	**COLLOID GOITRE**	**TOXIC GOITRE**	**CARCINOMA THYROID**
MALE	3	1	3	16
FEMALE	66	7	13	11

FIGURE-I

SEX DISTRIBUTION IN THE STUDY

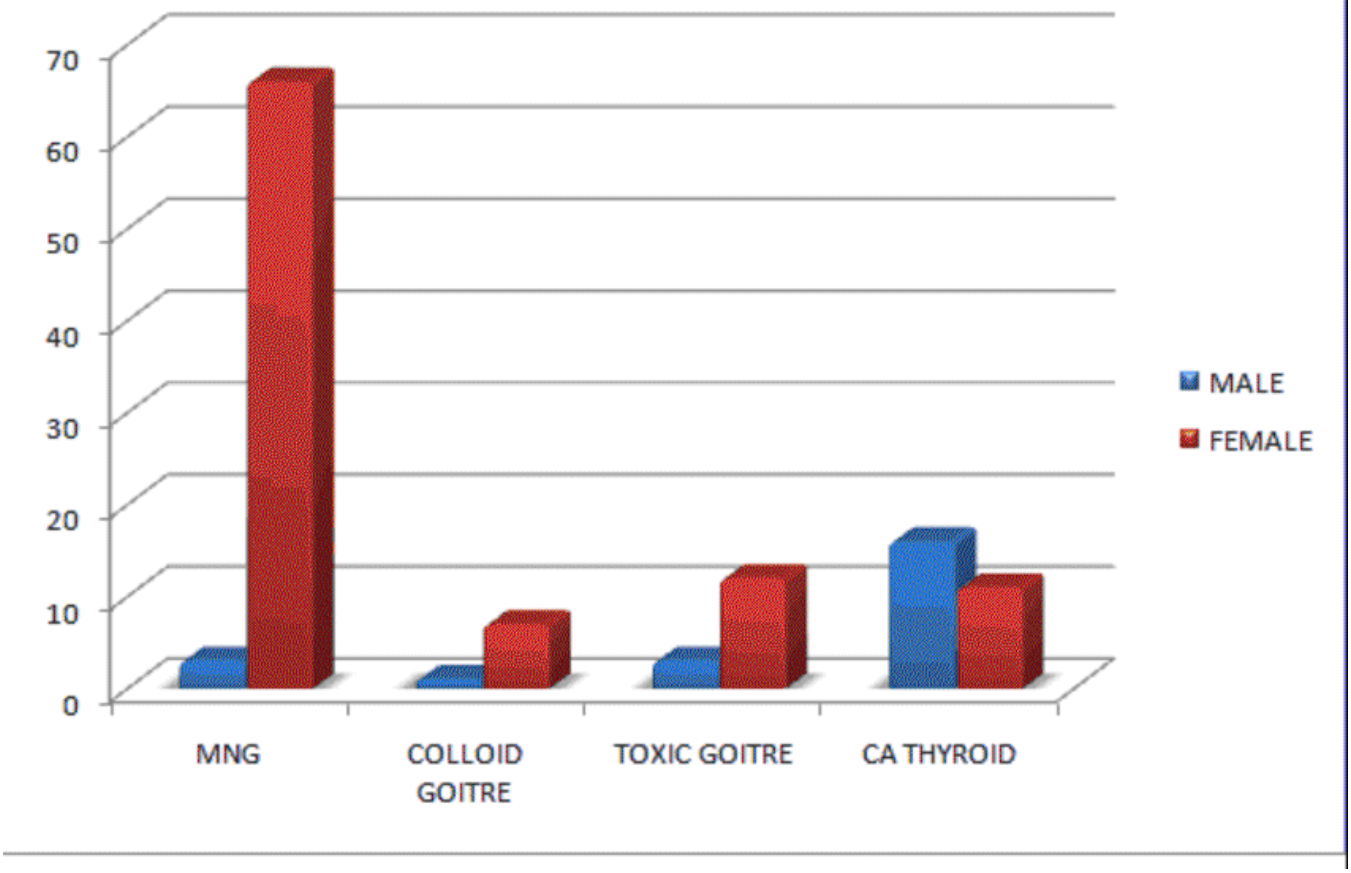


TABLE-IV

DITRIBUTION OF HYPOCALCEMIA AMONG AGE GROUPS OF PATIENTS IN THE STUDY.

AGE(YEARS)	HYPOCALCEMIA	
	TRANSIENT	PERMANENT
10-20	1	1
20-30	3	-
30-40	2	2
40-50	5	1
>50	15	5
TOTAL	26	9

TABLE-III

DISTRIBUTION OF HYPOCALCEMIA IN THE STUDY AMONG THE DIADNOSES & PROCEDURES PERFORMED.

	HYPOCALCEMIA	
	TRANSIENT	**PERMANENT**
DIAGNOSIS		
MULTINODULAR GOITRE	**8**	**1**
COLLOID GOITRE	**1**	**-**
TOXIC GOITRE	**2**	**2**
CARCINOMA THYROID	**15**	**6**
PROCEDURE		
NEAR-TOTAL THYROIDECTOMY	**11**	**2**
TOTAL THYROIDECTOMY	**6**	**2**
TOTALTHYROIDECTOMY WITH MRND	**9**	**5**
TOTAL	**26**	**9**

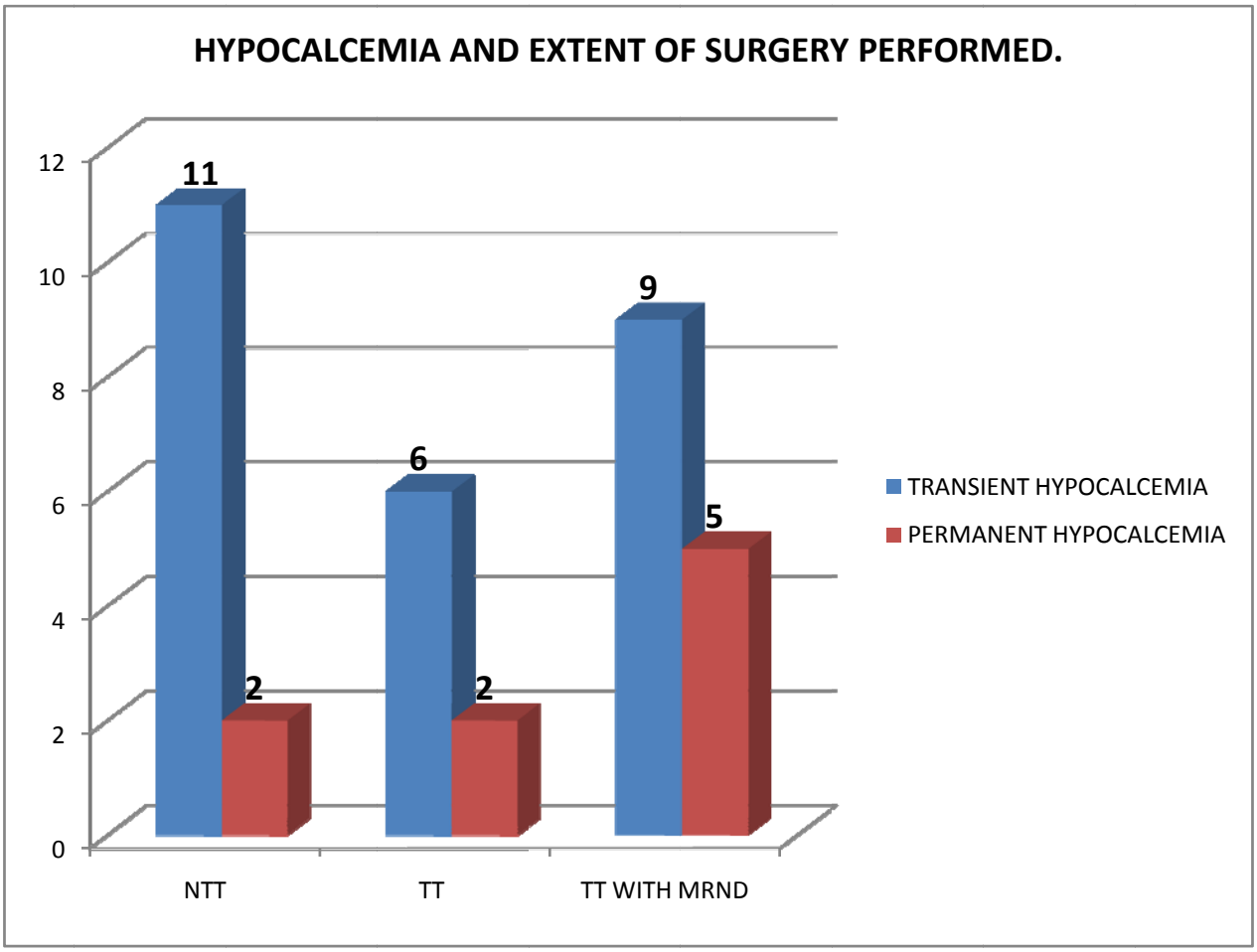

HYPOCALCEMIA AND EXTENT OF SURGERY PERFORMED.
12
10
8
6
4
2
0
11
2
6
2
9
5
NTT
TT
TT WITH MRND
TRANSIENT HYPOCALCEMIA
PERMANENT HYPOCALCEMIA

FIGURE-II

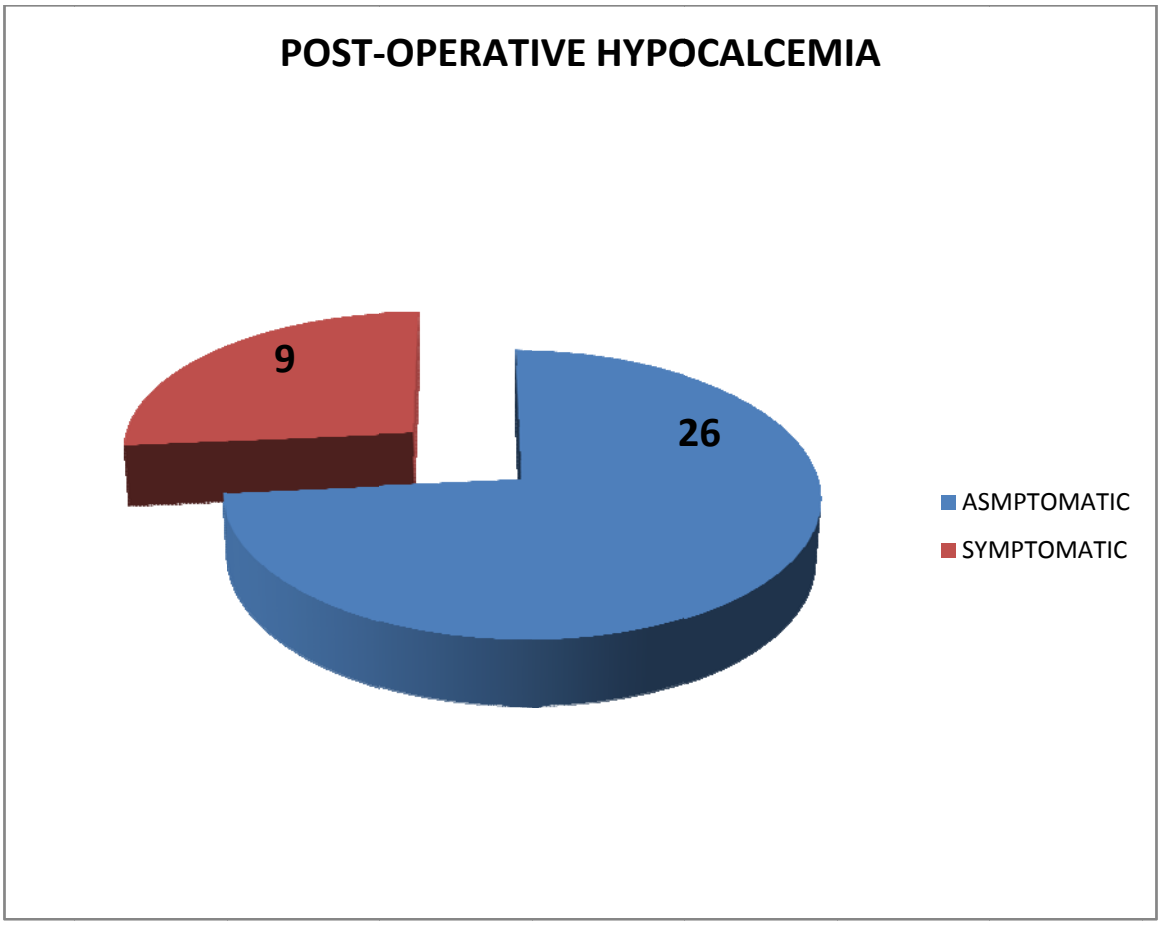

POST-OPERATIVE HYPOCALCEMIA
9
26
ASMPTOMATIC
SYMPTOMATIC

FIGURE-III

TABLE-V

DISTRIBUTION OF SIGNS AND SYMPTOMS OF 9 SYMPTOMATIC PATIENTS.

SIGNS&SYMPTOMS	PATIENTS
Chvostek	3
Paraesthesia&Numbness	5
Trousseau	3
Myalgias	5
Facial spasms	4
Carpal spasms	2
Pedal spasms	1

Total patients with hypocalcemia, n=26

Symptomatic patients=9 (34%)

DISCUSSION

In our study which is a prospective type 120 patients were included over a period of 2 years from January 2010 to December 2011.Regarding the sex distribution in this study, as per Table-II there were 23 males (19.2%) and 117 females(81.8%),thus we can infer that thyroid disorders are overwhelmingly more common in the fairer sex.But malignancy of thyroid gland was more common in male gender(60% of cases), probably male gender being in a greater risk for malignancy as per the AGES/AMES Criteria for thyroid malignancies.[7]

But the benign disorders of thyroid were more common among females in age group of 20-50 yrs(60%) as per Table-I. Malignant disorders of thyroid were more common after 50 yrs of age.

The **incidence of hypocalcemia in our study was 26 cases out of 120 operated (21.6%).**Out of the 26 cases that developed hypocalcemia in our study 9 patients(34.6%) went in for permanent hypocalcemia or hypoparathyroidism which is defined as hypocalcemia persisting after 6 months of surgery, so we can put it in another way by saying that 35% of

patients with transient hypocalcemia went in for permanent hypoparathyroidism requiring lifelong calcium and/or vitamin D3 supplementation.

So the **incidence of permanent hypocalcemia in our study is 7.5%.**

Regarding the distribution of hypocalcemia in our study (Table-III) ,there is a greater risk of developing hypocalcemia after malignant disorders of thyroid (58% of hypocalcemic pts). Also the rate of conversion to permanent hypocalcemia is 40% in malignancy of thyroid(6 out of 15 patients went in for permanent hypocalcemia after surgery),while the incidence of permanent hypocalcemia is 3% in benign thyroid disorders,out of which 2 cases developed permanent hypocalcemia ,which was due to a Near-total thyroidectomy being performed for Toxic goiter and may be as a result of "Hungry bone syndrome" causing the post-operative hypocalcemia where serum calcium is sequestered in bone after the underlying pathology is removed..This can be explained by figure-II which shows the extent of surgery performed and the occurrence of hypocalcemia which is more after more extensive procedures like Total thyroidectomy with/without modified radical neck dissection.Though the incidence of hypocalcemia after Near Total Thyroidectomy is 11.5%(11 out of 96 operated cases),this was only a

transient post-operative hypocalcemia and only two of these patients went in for a permanent hypocalcemic/hypoparathyroid state which was after surgery for toxic goiter for the reasons mentioned before.

In addition the incidence of hypocalcemia following malignant thyroid disorders is more than 50% (62.5%; 15 out of 24 cases.) and out of which 6 patients (25%) went in for permanent hypocalcemia.

Finally as we can see from figure-III out of 26 cases of hypocalcemia recorded in our study only 9 patients(35%) were symptomatic. Among these 9 symptomatic patients we infer from Table-V that the most common symptom occurring in hypocalcemic patients is paraesthesias, numbness and myalgias(5 cases) followed by facial spasms(4 cases) and chvosteks and trousseau's sign (3 cases each).

The reported incidence of post-thyroidectomy hypocalcemia varies from 1.6% to 50% when we review literature[92,93], estimates of the incidence of post-thyroidectomy hypoparathyroidism vary widely, ranging from 6.9% to 46% for transient and 0.4% to 33% for permanent hypoparathyroidism.[86]

Chow et al reported 16% permanent hypocalcaemia after total thyroidectomy, an incidence, which reached to 30% when patients had

thyroid cancers. In 310 patients with total thyroidectomy in the Chaudhary series, 17 patients (5.48%) had hypocalcaemia, 12 patients transient, and 5 had permanent hypocalcaemia.[84,85]

A multicenter prospective trial by Thomusch et al of 5846 patients undergoing total thyroidectomy revealed an incidence of transient hypoparathyroidism of 7.3% and permanent hypoparathyroidism of 1.5%.[86] When we compare the statistics of these authors with ours (transient hypocalcemia-21.6%; permanent hypocalcemia-7.5%), our rates are on the slightly higher side which probably could be due to more extensive surgery performed in our series for more number of malignant disorders of thyroid in onr study.

As regard the underlying thyroid pathology, higher incidence of hypo-calcaemia with malignant (25%) and Toxic goitre (11.4%) than that in simple nodular goiter (3.6%) the high incidence of hypocalcaemia in thyro-toxicosis was noted also by Wingert et al reported an incidence of 59% for hypocalcaemia after thyroidectomy for thyrotoxicosis versus 3% incidence after thyroidectomy for simple non toxic goiter.[94]. Many theories developed to explain this high

incidence of hypocalcaemia after thyroidectomy for thyrotoxicosis, Golding and Krane noted that in thyrotoxicosis there is increased bone catabolism and it has been demonstrated that the degree of thyrotoxic osteodystrophy increases proportionally with the severity of thyrotoxicosis. Michie et al postulated that the presence of osteodystrophy that excisted after surgery that was called "Hungery bone syndrome" is a possible explanation.[95]

In the Ganecalves series of total thyroidectomy for thyroid cancer, transient hypocalcaemia was reported in 25.5% of cases and permanent hypocalcaemia in 5.1%; when radical neck dissection was added to total thyroidectomies, these rates increased.[87], on comparison with our series our values are higher (transient hypocalcemia-62.5%; permanent hypocalcemia-25%%) again probably due to more frequency of concomitant radical neck dissections performed along with a total thyroidectomy in our series.

According to a study by Vayesberg et al , skill and experience of surgeons in performing total thyroidectomies were evaluated and hypocalcaemia was observed in 6.3% of the patients ,the incidence being inversely proportional to skill and experience of surgeon.[88]

However a survey by the American College of Surgeons reported an incidence of hypoparathyroidism following total thyroidectomy as 8%.[89]In our study the reported incidence following total thyroidectomy is 28% again due to the more extensive procedures performed (neck dissections combined with total thyroidectomy) and due to the absence of utilisation auto-transplantation of parathyroids in our study.

Finally permanent hypoparathyroidism is slightly more frequent in cases of thyroidectomy for hyperthyroidism, thyroid carcinoma, or after previous neck surgery—three conditions in which parathyroid preservation may be jeopardized by technical difficulties. Indeed, the number of parathyroid glands preserved during thyroidectomy appeared to be the major determinant of the outcome.

CONCLUSION

Postoperative hypocalcemia is the most common and sometimes the most severe and potentially debilitating complication observed after Near total thyroidectomy and Total thyroidectomy.

The incidence of hypocalcemia(transient) in our study is 21.6% and permanent hypocalcemia requiring lifelong calcium and/or vitamin D3 supplementation is 7.5%.

So proper planning and meticulous surgical technique with special emphasis in localization and preserving the vascularity of atleast one para-thyroid gland will go a long way in reducing this complication.

We should employ newer techniques like auto-transplantation of parathyroids, use of Ultra sonic shears and enhanced bipolar diathermy which may help in bringing down the incidence of post-operative hypocalcemia.

We can avoid Transient/Permanent hypocalcemia or hypoparathyroidism by searching for parathyroid glands in the post-operative specimen, if detected immediate auto-transplantation of parathyroids will reduce the incidence of transient/permanent hypocalcemia following thyroidectomies.

Proper post-operative monitoring and early detection by performing a serum calcium assay and even an intact-iPTH assay(if available and affordable) is mandatory to prevent post-operative distress to patients.

Adequate and timely calcium and /or vitamin D3 supplementation is advised in patients at risk for developing post-operative hypocalcemia and for treatment of all symptomatic patients.

Finally a proper follow-up schedule is mandatory in all patients undergoing Near-Total Thyroidectomy and Total Thyroidectomy.

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PROFORMA

NAME OF PATIENT :

AGE/SEX :

IP NUMBER :

ADDRESS & CONTACT NO :

DATE OF ADMISSION :

DATE OF SURGERY :

DATE OF DISCHARGE :

DIAGNOSIS :

SURGERY DONE :

INVESTIGATIONS

TFT : T_3= , T_4= , TSH=

FNAC :

ULTRASOUND NECK :

SERUM CALCIUM

- IMMEDIATE POST OP PERIOD :
- AFTER ONE MONTH :
- AFTER SIX MONTHS :

ANY SIGNS & SYMPTOMS OF HYP0CALCEMIA

- IMMEDIATE POST OP PERIOD :
- AFTER ONE MONTH :
- AFTER SIX MONTHS :

MASTER CHART

SL.NO:	IP NO:	AGE	SEX	DIAGNOSIS	PROCEDURE	HYPOCALCEMIA	
						TRANSIENT	PERMANENT
1.	095573	50	F	MNG	NTT	N	N
2.	092940	40	F	CA THYROID	TT	N	N
3.	097556	35	F	COLLOID GOITRE	NTT	N	N
4.	103316	25	F	CA THYROID	TT WITH RT.MRND	P	N
5.	107092	26	F	TOXIC MNG	NTT	P	N
6.	107080	43	F	MNG	NTT	N	N
7.	004383	18	F	MNG	NTT	N	N
8.	004388	20	M	CA THYROID	TT WITH LT. FND	N	N
9.	013760	28	F	MNG	NTT	N	N
10.	017324	50	F	CA THYROID	TT	N	N
11.	018241	50	F	TOXIC MNG	NTT	N	N
12.	033321	51	F	MNG	NTT	N	N
13.	035611	36	F	MNG	NTT	N	N
14.	036658	22	F	MNG	NTT	N	N
15.	O40546	38	M	MNG	NTT	N	N
16.	044425	44	F	MNG	NTT	N	N
17.	052339	50	F	MNG	NTT	N	N
18.	054169	35	M	CA THYROID	TT WITH RT.MRND	P	P
19.	050362	43	M	TOXIC MNG	NTT	N	N

20.	060211	45	F	MNG	NTT	N	N
21.	065787	14	F	MNG	NTT	N	N
22.	069712	40	F	MNG	NTT	P	N
23.	063928	40	F	TOXIC MNG	NTT	N	N
24.	050383	27	F	CA THYROID	TT	P	N
25.	072238	62	M	TOXIC MNG	NTT	N	N
26.	073902	42	F	TOXIC MNG	NTT	N	N
27.	075537	59	F	CA THYROID	TT WITH RT.MRND	N	N
28.	084974	48	M	CA THYROID	TT	N	N
29.	085066	38	F	TOXIC MNG	NTT	N	N
30.	088048	70	F	MNG	NTT	N	N
31.	092620	37	F	MNG	NTT	N	N
32.	000480	42	M	CA THYROID	TT WITH BIL.MRND	P	P
33.	004290	44	F	MNG	NTT	N	N
34.	005167	23	F	MNG	NTT	N	N
35.	008366	55	F	TOXIC MNG	NTT	P	P
36.	009964	52	F	MNG	NTT	N	N
37.	014799	48	F	CA THYROID	TT	N	N
38.	014783	30	F	MNG	NTT	N	N
39.	017975	48	F	MNG	NTT	N	N
40.	013183	31	F	TOXIC MNG	NTT	N	N

SL.NO:	IP NO:	AGE	SEX	DIAGNOSIS	PROCEDURE	HYPOCALCEMIA	
						TRANSIENT	PERMANENT
41.	024044	26	F	MNG	NTT	N	N
42.	025637	39	F	MNG	NTT	N	N
43.	027371	15	F	MNG	NTT	N	N
44.	025712	50	F	TOXIC MNG	NTT	P	P
45	021070	36	M	CA THYROID	TT	P	N
46.	025724	32	F	CA THYROID	TT WITH RT. MRND	P	P
47.	033428	57	F	CA THYROID	TT	N	N
48.	032275	41	F	MNG	NTT	N	N
49.	032404	48	F	MNG	NTT	P	N
50.	314472	45	F	TOXIC MNG	NTT	N	N
51.	030575	37	F	TOXIC MNG	NTT	N	N
52.	033952	50	F	MNG	NTT	N	N
53.	040454	46	F	MNG	NTT	N	N
54.	038830	40	F	MNG	NTT	N	N
55.	043836	40	F	MNG	NTT	N	N
56.	042401	42	M	CA THYROID	TT WITH RT.MRND	P	N
57.	045488	55	F	MNG	NTT	N	N
58.	045591	29	F	MNG	NTT	N	N
59.	040143	38	F	COLLOID GOTRE	TT	N	N

60.	041345	42	F	MNG	NTT	N	N
61.	046123	17	F	COLLOID GOITER	NTT	N	N
62.	056321	25	F	MNG	NTT	N	N
63.	056361	35	F	MNG	NTT	N	N
64	057234	38	F	MNG	NTT	N	N
65	057452	29	F	MNG	NTT	N	N
66	058312	37	F	MNG	NTT	N	N
67	058567	52	F	MNG	NTT	N	N
68	059321	60	F	MNG	NTT	P	N
69	059679	47	F	MNG	NTT	N	N
70.	067452	53	F	MNG	NTT	N	N
71.	006834	38	F	MNG	NTT	N	N
72	078124	39	M	MNG	NTT	N	N
73.	079235	45	F	MNG	NTT	N	N
74.	079658	19	F	COLLOID GOITER	NTT	N	N
75.	082349	29	F	MNG	NTT	P	N
76.	085675	45	M	MNG	NTT	N	N
77	086789	42	F	MNG	NTT	N	N
78.	087821	52	F	MNG	NTT	N	N
79.	093216	38	F	MNG	NTT	P	N
80.	096591	50	F	MNG	NTT	N	N

SL.NO:	IP NO:	AGE	SEX	DIAGNOSIS	PROCEDURE	HYPOCALCEMIA	
						TRANSIENT	PERMANENT
81.	057279	60	F	COLLOID GOTER	TT	P	N
82.	045959	33	F	MNG	NTT	N	N
83.	045952	42	F	CA THYROID	TT	N	N
84.	074151	65	M	CA THYROID	TT WITH RT. MRND	N	N
85.	078044	37	F	CA THYROID	TT WITH BIL. MRND	P	P
86.	078105	50	F	CA THYROID	TT WITH BIL. MRND	P	P
87.	034329	32	F	TOXIC MNG	TT	P	N
88.	009122	47	F	MNG	NTT	N	N
89.	096481	35	F	MNG	NTT	N	N
90	100573	24	F	MNG	NTT	N	N
91	017418	28	F	MNG	TT	N	N
92.	033108	55	M	CA THYROID	TT	P	P
93.	043319	33	F	MNG	NTT	N	N
94	044275	32	F	MNG	NTT	N	N
95	046272	31	F	MNG	NTT	N	N
96	070251	25	F	MNG	NTT	N	N
97	078931	45	M	CA THYROID	TT WITH BIL. MRND	N	N
98	083481	35	F	MNG	NTT	N	N
99	086451	29	F	MNG	NTT	N	N

100.	098321	46	F	MNG	NTT	N	N
101.	083457	65	M	CA THYROID	TT WITH BIL. MRND	**P**	**N**
102.	079780	33	F	TOXIC MNG	TT	**N**	**N**
103.	083379	45	M	MNG	NTT	**N**	**N**
104	021070	36	M	CA THYROID	TT	**N**	**N**
105.	030579	65	M	CA THYROID	TT WITH LT. MRND	**P**	**N**
106.	015911	35	M	TOXIC MNG	TT	**N**	**N**
107.	054169	35	M	CA THYROID	TT	**P**	**N**
108	056789	47	F	MNG	NTT	**N**	**N**
109	057345	36	F	MNG	NTT	**N**	**N**
110.	065007	60	M	CA THYROID	TT	**P**	**N**
111.	015949	37	M	CA THYROID	TT WITH RT.MRND	**P**	**N**
112.	083379	45	F	MNG	NTT	**N**	**N**
113	085679	36	F	MNG	NTT	**N**	**N**
114	086123	18	F	COLLOID GOITER	NTT	**N**	**N**
115	087345	29	F	MNG	NTT	**N**	**N**
116.	088122	43	F	MNG	NTT	**P**	**N**
177.	041790	65	M	CA THYROID	TT	**N**	**N**
118.	095671	45	F	COLLOID GOITER	NTT	**P**	**N**
119.	109872	38	F	MNG	NTT	**N**	**N**
120.	039479	22	F	MNG	NTT	**N**	**N**

KEY TO MASTER CHART

DIAGNOSIS

MNG = MULTI NODULAR GOITER

PROCEDURE

NTT = NEAR TOTAL THYROIDECTOMY

TT = TOTAL THYROIDECTOMY

MRND = MODIFIED RADICAL NECK DISSECTION

FND = FUNCTIONAL NECK DISSECTION

HYPOCALCEMIA

N = NOT PRESENT

P = PRESENT